

Cadmium Compounds [\(A\)](#)

Hazard Summary-Created in April 1992; Revised in January 2000

The main sources of cadmium in the air are the burning of fossil fuels such as coal or oil and the incineration of municipal waste. The acute (short-term) effects of cadmium in humans through inhalation exposure consist mainly of effects on the lung, such as pulmonary irritation. Chronic (long-term) inhalation or oral exposure to cadmium leads to a build-up of cadmium in the kidneys that can cause kidney disease. Cadmium has been shown to be a developmental toxicant in animals, resulting in fetal malformations and other effects, but no conclusive evidence exists in humans. An association between cadmium exposure and an increased risk of lung cancer has been reported from human studies, but these studies are inconclusive due to confounding factors. Animal studies have demonstrated an increase in lung cancer from long-term inhalation exposure to cadmium. EPA has classified cadmium as a Group B1, probable human carcinogen.

Please Note: The main sources of information for this fact sheet are EPA's [Integrated Risk Information System \(IRIS\)](#), which contains information on oral chronic toxicity and the [RfD](#), and the carcinogenic effects of cadmium including the unit cancer risk for inhalation exposure, and the Agency for Toxic Substances and Disease Registry's (ATSDR's) [Toxicological Profile for Cadmium](#).

Uses

- Most cadmium used in the United States today is obtained as a byproduct from the smelting of zinc, lead, or copper ores. [\(1\)](#)
Cadmium is used to manufacture pigments and batteries and in the metal-plating and plastics industries. [\(1\)](#)

Sources and Potential Exposure

- The largest sources of airborne cadmium in the environment are the burning of fossil fuels such as coal or oil, and incineration of municipal waste materials. Cadmium may also be emitted into the air from zinc, lead, or copper smelters. [\(1\)](#)
- For nonsmokers, food is generally the largest source of cadmium exposure. Cadmium levels in some foods can be increased by the application of phosphate fertilizers or sewage sludge to farm fields. [\(1\)](#)
- Smoking is another important source of cadmium exposure. Smokers have about twice as much cadmium in their bodies as do nonsmokers. [\(1\)](#)

Assessing Personal Exposure

- The amount of cadmium present in blood or urine can be measured by atomic absorption spectrophotometry and used as an indication of cadmium exposure. [\(1\)](#)
- A more precise method, called neutron activation analysis, can be used to measure cadmium concentrations in the liver or kidney. [\(1\)](#)

Health Hazard Information

Acute Effects:

- Acute inhalation exposure to high levels of cadmium in humans may result in effects on the lung, such as bronchial and pulmonary irritation. A single acute exposure to high levels of cadmium can result in long-lasting impairment of lung function. [\(1,3,4\)](#)
- Cadmium is considered to have [high](#) acute toxicity, based on short-term animal tests in rats. [\(5\)](#)

Chronic Effects (Noncancer):

- Chronic inhalation and oral exposure of humans to cadmium results in a build-up of cadmium in the kidneys that can cause kidney disease, including proteinuria, a decrease in glomerular filtration rate, and an increased frequency of kidney stone formation. [\(1,3,4\)](#)

- Other effects noted in occupational settings from chronic exposure of humans to cadmium in air are effects on the lung, including bronchiolitis and emphysema. ([1,3,4](#))
- Chronic inhalation or oral exposure of animals to cadmium results in effects on the kidney, liver, lung, bone, immune system, blood, and nervous system. ([1,3](#))
- The Reference Dose ([RfD](#)) for cadmium in drinking water is 0.0005 milligrams per kilogram per day (mg/kg/d) and the [RfD](#) for dietary exposure to cadmium is 0.001 mg/kg/d; both are based on significant proteinuria in humans. The [RfD](#) is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily oral exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious noncancer effects during a lifetime. It is not a direct estimator of risk, but rather a reference point to gauge the potential effects. At exposures increasingly greater than the [RfD](#), the potential for adverse health effects increases. Lifetime exposure above the [RfD](#) does not imply that an adverse health effect would necessarily occur. ([6](#))
- EPA has high confidence in both [RfDs](#) based primarily on a strong database for cadmium toxicity in humans and animals that also permits calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism, and elimination. ([6](#))
- EPA has not established a Reference Concentration ([RfC](#)) for cadmium. ([6](#))
- The [California Environmental Protection Agency](#) (CalEPA) has established a chronic reference exposure level of 0.00001 milligrams per cubic meter (mg/m³) for cadmium based on kidney and respiratory effects in humans. The CalEPA reference exposure level is a concentration at or below which adverse health effects are not likely to occur. ([7](#))

Reproductive/Developmental Effects:

- Limited evidence exists for an association between inhalation exposure and a reduction in sperm number and viability in humans. ([1](#))
- Human developmental studies on cadmium are limited, although there is some evidence to suggest that maternal cadmium exposure may result in decreased birthweights. ([1](#))
- Animal studies provide evidence that cadmium has developmental effects, such as low fetal weight, skeletal malformations, interference with fetal metabolism, and impaired neurological development, via inhalation and oral exposure. ([1,3,4](#))
- Limited animal data are available, although some reproductive effects, such as decreased reproduction and testicular damage, have been noted following oral exposures. ([1](#))

Cancer Risk:

- Several occupational studies have reported an excess risk of lung cancer in humans from exposure to inhaled cadmium. However, the evidence is limited rather than conclusive due to confounding factors. ([1,3,6](#))
- Animal studies have reported cancer resulting from inhalation exposure to several forms of cadmium, while animal ingestion studies have not demonstrated cancer resulting from exposure to cadmium compounds. ([1,3,6](#))
- EPA considers cadmium to be a probable human carcinogen (cancer-causing agent) and has classified it as a Group B1 carcinogen. ([6](#))
- EPA uses mathematical models, based on animal studies, to estimate the probability of a person developing cancer from breathing air containing a specified concentration of a chemical. EPA calculated an inhalation unit risk estimate of $1.8 \times 10^{-3}(\mu\text{g}/\text{m}^3)^{-1}$. EPA estimates that, if an individual were to continuously breathe air containing cadmium at an average of $0.0006 \mu\text{g}/\text{m}^3$ ($6 \times 10^{-7} \text{ mg}/\text{m}^3$) over his or her entire lifetime, that person would theoretically have no more than a one-in-a-million increased chance of developing cancer as a direct result of breathing air containing this chemical. Similarly, EPA estimates that continuously breathing air containing $0.006 \mu\text{g}/\text{m}^3$ ($6 \times 10^{-6} \text{ mg}/\text{m}^3$) would result in not greater than a one-in-a-hundred thousand increased chance of developing cancer, and air containing $0.06 \mu\text{g}/\text{m}^3$ ($6 \times 10^{-5} \text{ mg}/\text{m}^3$) would result in not greater than a one-in-ten thousand increased chance of developing cancer. For a detailed discussion of confidence in the potency estimates, please see IRIS. ([6](#))

Physical Properties

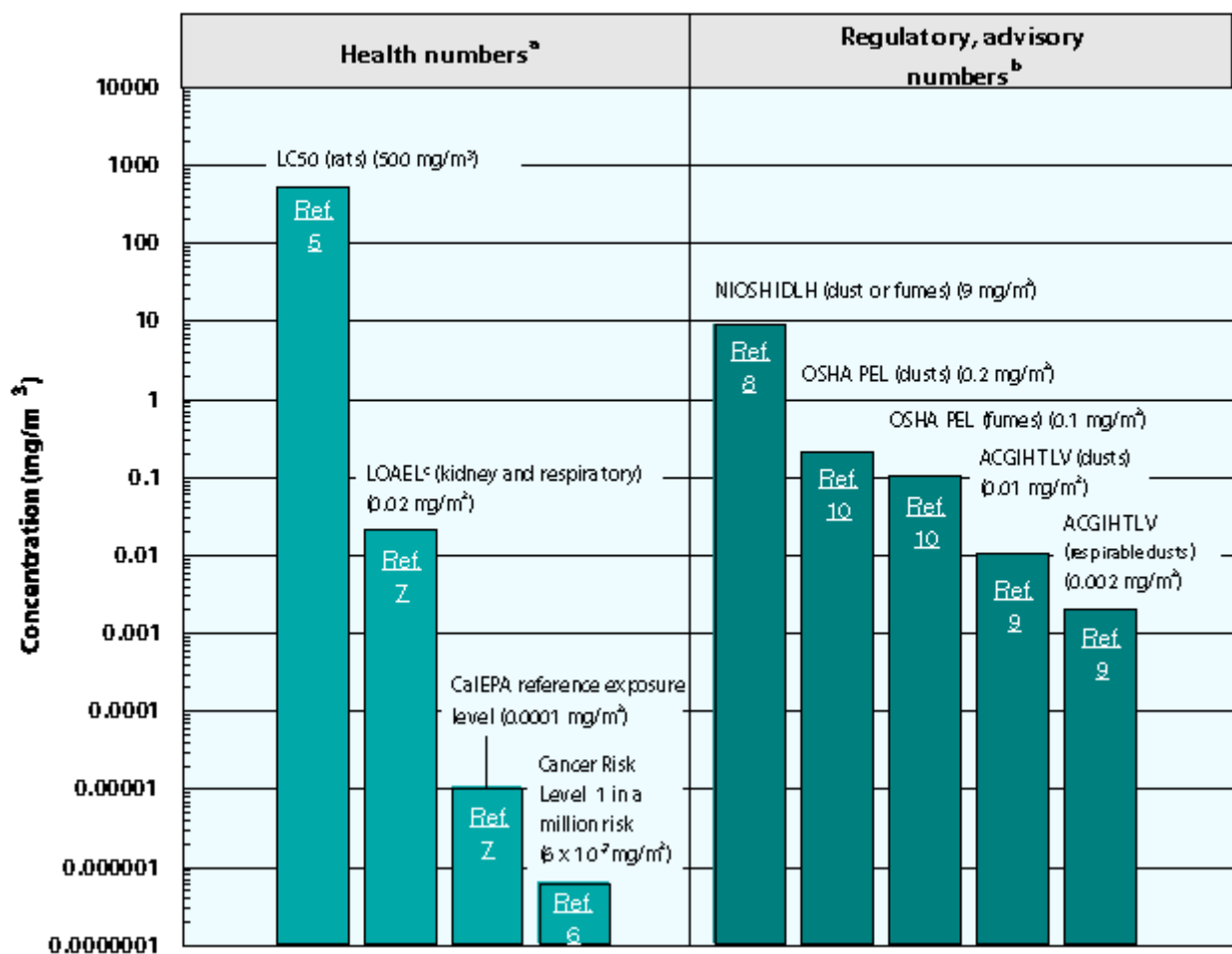
- Cadmium is a soft silver-white metal that is usually found in combination with other elements. ([1](#))
 - Cadmium compounds range in solubility in water from quite soluble to practically insoluble. ([1](#))
 - The chemical symbol for cadmium is Cd and the atomic weight is 112.41 g/mol. ([1](#))
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Conversion Factors (only for the gaseous form):

To convert concentrations in air (at 25°C) from ppm to mg/m³: $\text{mg/m}^3 = (\text{ppm}) \times (\text{molecular weight of the compound}) / (24.45)$. For cadmium: 1 ppm = 4.6 mg/m³. To convert concentrations in air from µg/m³ to mg/m³: $\text{mg/m}^3 = (\mu\text{g/m}^3) \times (1 \text{ mg}/1000 \mu\text{g})$.

Health Data from Inhalation Exposure

Cadmium



ACGIH TLV--American Conference of Governmental and Industrial Hygienists' threshold limit value expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effects.

LC₅₀ (Lethal Concentration₅₀)--A calculated concentration of a chemical in air to which exposure for a specific length of time is expected to cause death in 50% of a defined experimental animal population.

NIOSH IDLH--National Institute of Occupational Safety and Health's immediately dangerous to life and health; NIOSH concentration representing the maximum level of a pollutant from which an individual could escape within 30 minutes without escape-impairing symptoms or irreversible health effects.

OSHA PEL--Occupational Safety and Health Administration's permissible exposure limit expressed as a time-weighted average; the concentration of a substance to which most workers can be exposed without adverse effect averaged over a normal 8-h workday or a 40-h workweek.

The health and regulatory values cited in this factsheet were obtained in December 1999.

^aHealth numbers are toxicological numbers from animal testing or risk assessment values developed by EPA.

^bRegulatory numbers are values that have been incorporated in Government regulations, while advisory numbers are nonregulatory values provided by the Government or other groups as advice. OSHA numbers are regulatory, whereas NIOSH and ACGIH numbers are advisory.

^cThe LOAEL is from the critical study used as the basis for the CalEPA chronic reference exposure level.

References

1. Agency for Toxic Substances and Disease Registry (ATSDR). *Toxicological Profile for Cadmium*. Draft for Public Comment. Public Health Service, U.S. Department of Health and Human Services, Atlanta, GA. 1997.
2. U.S. Environmental Protection Agency. *Deposition of Air Pollutants to the Great Waters. First Report to Congress*. EPA-453/R-93-055. Office of Air Quality Planning and Standards, Research Triangle Park, NC. 1994.
3. E.J. Calabrese and E.M. Kenyon. *Air Toxics and Risk Assessment*. Lewis Publishers, Chelsea, MI. 1991.
4. U.S. Department of Health and Human Services. Hazardous Substances Data Bank ([HSDB, online database](#)). National Toxicology Information Program, National Library of Medicine, Bethesda, MD. 1993.
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6. U.S. Environmental Protection Agency. [Integrated Risk Information System \(IRIS\) on Cadmium](#). National Center for Environmental Assessment, Office of Research and Development, Washington, DC. 1999.
7. [California Environmental Protection Agency \(CalEPA\)](#). *Technical Support Document for the Determination of Noncancer Chronic Reference Exposure Levels. Draft for Public Comment*. Office of Environmental Health Hazard Assessment, Berkeley, CA. 1997.
8. National Institute for Occupational Safety and Health (NIOSH). [Pocket Guide to Chemical Hazards](#). U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention. Cincinnati, OH. 1997.
9. American Conference of Governmental Industrial Hygienists (ACGIH). *1999 TLVs and BEIs. Threshold Limit Values for Chemical Substances and Physical Agents. Biological Exposure Indices*. Cincinnati, OH. 1999.
10. Occupational Safety and Health Administration (OSHA). Occupational Safety and Health Standards, Toxic and Hazardous Substances. *Code of Federal Regulations*. 29 CFR 1910.1000. 1998.

A. * This fact sheet discusses cadmium and cadmium compounds. Most of the information is on cadmium, except in those cases where there are differences in toxicity between cadmium and cadmium compounds. In these cases, information on the cadmium compound is presented.